Vitiligo
Definition

Vitiligo is a circumscribed, acquired, idiopathic, progressive hypomelanosis of skin and hair which is often familial and is characterized microscopically by an absence of melanocytes.

Leukoderma is the term applied only to depigmented patches of known causes eg: following burns, chemicals, inflammatory disorder.
Normal Skin Color

- Melanin
- Carotenoids

Epidermal

- Oxyhaemoglobin
- Reduced haemoglobin

Dermal
Normal Melanisation

Melanocyte

Neuroectodermal Origin

Migrates to

Cutaneous

Epidermal

Mucous membrane

Appendageal

Follicular

Non follicular

Extracutaneous

Eye

Brain
Normal Melanisation

Epidermal melanocyte

- Attached to basement membrane
- Rarely divide
- Require bFGF (Basic fibroblast growth factor) for growth and multiplication
- Function - Endogenous sunscreen
- Response to injury - Unpredictable
Hair melanocyte

Hair follicle melanocyte

Hair bulb

• Dendritic
• Functional

Mid-follicle + Upper follicle

• DOPA Negative
• Amelanotic & Non dendritic
• Active after trauma
Normal Melanisation

Melanosome

- Membrane bound melanosome inside the melanocyte
- Site of production and storage of melanin
- Membrane prevents diffusion of intermediate toxic products of melanin synthesis which are harmful to melanocyte
Aetiopathogenesis

Vitiligo

Melanocytopenia

Pathogenesis

End organ disease

Secondary to

• Auto antibodies
• Neural secretion
Aetiopathogenesis

- End organ disease
  Apoptosis, Self destruction of melanocyte
  Cause: ↑ amount, diffusion of intermediate products
  ↑ oxidative stress
- Autoimmune
  Vitiligo antigen: Vit 40, Vit 75, Vit 90
  Epidermal melanocytes express more vitiligo antigen than hair follicle melanocyte
- Neural
  Nerve endings maybe secreting toxic substances which is detrimental to melanocyte
Clinical features of Vitiligo

Macule of Vitiligo:

- Round, oval
  - Milky white
  - Scalloped margin

- Trichrome or quadrichrome
- Confetti macules
- Inflammatory border in some cases
- Leucotrichia in some cases
Clinical Classification

- Localized
  - Focal
  - Segmental

- Generalized
  - Symmetrical
  - Acromucosal
  - Universalis
Cutaneous associations in Vitiligo

- Leucotrichia
- Premature gray hair
- Halo nevi
- Alopecia areata
Systemic associations in Vitiligo

- Thyroid disease
- Diabetes
- Addison's disease
- Pernicious anemia
- Multiple endocrinopathy syndrome
Differential diagnosis

- Piebaldism
- Pityriasis Alba
- Hansens disease
- Pityriasis Versicolor
- Morphoea
- Lichen Sclerosus et Atrophicus
- Post inflammatory leucoderma
Treatment guidelines

Vitiligo is a sign and the cause of melanocyte destruction may not be the same in every case. There is no uniform response to treatment.
Aims of treatment

- Repigmentation
- Prevention of further depigmentation
To increase melanin

Options:

- Increase number of melanocytes by promoting migration from hair follicle.
- Activate dormant melanocyte
- Increase production of melanin from existing functional melanocyte
Medical Treatment

- Psoralen + UVA
- UVB – Narrowband
- Steroids
- Eau de Cologne
- Khellin + UVA
- L-phenylalanine + UVA
- Topical Tacrolimus
- Topical 5 Fluorouracil
- Topical Calcipotriol
- Placental extract
- Topical bFGF
- Excimer laser
Treatment options for repigmentation

- Topical steroids - All types of vitiligo
- Topical PUVA - Focal / segmental
- Systemic PUVA - Segmental / Generalized
Prevention of further depigmentation

- Treatment of precipitating cause
- Steroid
  - Topical (useful for repigmentation also)
  - Systemic
    - Oral
      - Short course
      - Pulse
    - Injectable
      - ACTH
      - Triamcinolone
PUVA (psoralen + UVA) therapy

- Drug + light
- Systemic/ Topical
  Psoralen + UVA (320-400nm)
  Trimethoxypsoralen, 8-methoxypsoralen
  UVA chamber, PUVASOL
  Photometer to measure output
  Protective goggles
Cutaneous response after PUVA therapy

- Erythema
- Perifollicular pigmentation
- Inhibition of cell proliferation
- Rarely oedema and vesiculation
Repigmentation
Treatment Protocol

TMP 0.6 mg/kg – 25 sittings
- No change

TMP 0.9 mg/kg – 25 sittings
- No change

8 MOP 0.3 mg/kg – 25 sittings
- No change

8 MOP 0.6 mg/kg – 25 sittings
- No change

TMP + 8MOP – 25 sittings
- No response
- Unresponsive case
PUVA Therapy

Follow up

Good response

Continue maintenance PUVA

Development of new patches

Increase dose

Total pigmentation

Stop PUVA
## PUVA Therapy: Side Effects

<table>
<thead>
<tr>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythema</td>
<td>Chronic actinic damage</td>
</tr>
<tr>
<td>Pruritus</td>
<td>Carcinoma - rare</td>
</tr>
<tr>
<td>Nausea</td>
<td>Immunosuppression</td>
</tr>
<tr>
<td>Headache</td>
<td>Ophthalmic effect</td>
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<tr>
<td>Koebner phenomenon</td>
<td>Premature cataract</td>
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</tbody>
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Topical steroids

- Isolated macules
  - Hydrocortisone
  - Mometasone
  - Betamethasone
  - Clobetasol

- Side effects
  - Atrophy
  - Striae

Children + Face

Adults + Body
Systemic steroids

- Low dose, long term
  - Oral
  - Injectable
- High dose pulse
- ACTH
Permanent depigmentation

- More than 50% area involvement
- Failure of treatment or does not wish to continue treatment
- 20% MBEH (monobenzyl ether of hydroquinone) – 4 to 12 months
- Irreversible
- Eyes, hair spared
- Needs sunscreen afterwards
- Side effect - contact dermatitis
- Rarely accepted by Indian patients
Prognostic factors

Cases resistant to medical line of treatment

- Acrofacial
- Patches on bony prominences
- Lesions on glans penis, palms, soles
- Patches with gray hair
- Patches around nipple
- Long standing cases
- Extensive depigmentation
Failure to respond to medical line of treatment indicates melanocyte reservoir is no more available in that area and it is needed to repopulate that area with melanocytes which can be achieved by various surgical modalities.
Surgical treatment of vitiligo

- Tattooing
- Dermabrasion
- Excision and closure
- Needling & spot peeling
- Punch grafting
- Split thickness grafting
- Suction blister grafting
- Melanocyte grafting
- Mesh grafting
- Allograft
Thank you